

CASE REPORT

Concrete induced cardiac contusion

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Abstract

A previously fit 22 year old man was struck in the chest by a concrete block dropped through the windscreen of his car while he was driving on the motorway. He suffered extensive chest wall trauma and lung contusion, which subsequently precipitated acute respiratory distress. On admission ECG showed right bundle branch block and left axis deviation. Three days later QRS duration was normal but there was anterior ST segment elevation and subsequent T wave change. There was a large rise in creatine kinase, and echocardiography revealed septal and apical hypokinesis as well as a mobile mass attached to the left side of the interventricular septum, which had the echogenic texture of myocardium. The patient had fixed perfusion defects in the areas of hypokinesis on thallium scanning but the coronary arteries were unobstructed at angiography. He was treated with warfarin in the short term and an angiotensin converting enzyme inhibitor in the longer term and has made an asymptomatic recovery. Outpatient echocardiography two months after the injury demonstrated some recovery in overall left ventricular systolic function and no evidence of the intracardiac mass. This case illustrates some of the typical features of non-fatal cardiac contusion associated with non-penetrating cardiac trauma, and was complicated by partial thickness avulsion of a strip of the myocardium in the interventricular septum.

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A previously fit 22 year old man was driving along a motorway when the windscreen of his car was smashed by a block of concrete that was dropped from the bridge above. He was struck in the chest by the missile, but managed to slow his car down and stop safely before being taken to the nearest accident and emergency department. On arrival he was con-

scious and neurologically intact. He was in respiratory distress with tachypnoea and a flail segment on the left side of his chest with tracheal deviation to the right. His pulse was 110 beats/min and blood pressure 110/70 mm Hg. Heart sounds were normal without added sounds, and all pulses were intact. Chest radiography demonstrated multiple rib fractures on the left with a pneumothorax on that side, as well as bilateral consolidation and surgical emphysema. Cervical spine radiography and computed tomography of the brain were normal. Arterial blood gases confirmed hypoxaemia and low PaCO₂. Admission ECG showed sinus rhythm, left axis deviation, and right bundle branch block (fig 1A). The patient was sedated, paralysed, and mechanically ventilated after a left chest drain was inserted. A transthoracic echocardiogram on admission to intensive care demonstrated normal left ventricular dimensions but with hypokinesis of the septum and apical areas. No pericardial effusion was present and no valve abnormalities or intracardiac masses were noted.

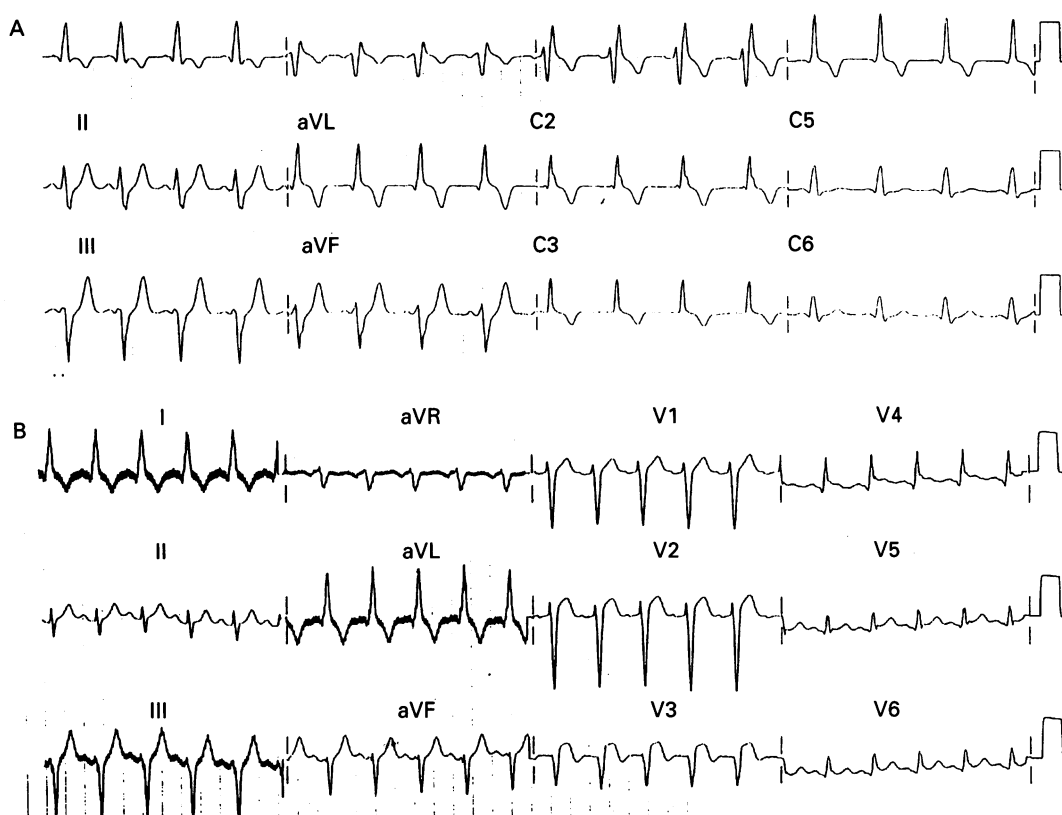
The patient required aggressive "volume recruitment" ventilation using pressure controlled inverse ratio ventilation with high inspired oxygen concentrations for increasing hypoxaemia. On day 3 after admission his ECG exhibited significant ST segment elevation in the anterior leads, the QRS duration having returned to normal (fig 1B). Seven days after admission he was transferred to this centre because of continuing problems with ventilation related to acute lung injury as well as concern over the evolution of the anterior ECG changes. On arrival, computed tomography of the thorax showed persistent left pneumothorax and bilateral "ground glass" consolidation consistent, together with the hypoxaemia, with a diagnosis of acute respiratory distress syndrome. The ECG continued to show anterior ST segment elevation with biphasic T waves. Creatine kinase was 4853 U/l (normal < 170) and aspartate aminotransferase 84 U/l (normal 41). Transthoracic echocardiogram provided poor views, but the left ventricular dimensions were normal. Transoesophageal echocardiography revealed reduced contractility of the septum and apex,

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Figure 1 (A) ECG on admission to the accident and emergency department after original incident showing sinus rhythm, left axis deviation, and right bundle branch block. (B) ECG three days later showing anterior ST segment elevation with loss of anterior R wave, T wave inversion, and normal QRS deviation.



and anatomically normal valves. There was no evidence of aortic dissection and flow was seen in both proximal coronary arteries. A 1.5×2 cm mass was apparent on the left side of the interventricular septum that had the same echogenic texture as the surrounding myocardium; it was attached to the septum by a short stalk (fig 2). The end of this mass moved within the left ventricular cavity but there was no evidence of an intracardiac shunt. In view of the presence of this mass and the moderately impaired left ventricular function, warfarin and an angiotensin converting enzyme inhibitor were started.

By the 14th day after admission it was possible to wean the patient off the ventilator. A cardiac magnetic resonance imaging scan con-

firmed extensive apical and inferior septal hypokinesis (ejection fraction 32%) but did not demonstrate an intracardiac mass. A thallium radioisotope scan on day 19 revealed fixed perfusion defects in the apex and inferior septum, but there was no evidence of reversibility of perfusion after stress. Finally, coronary angiography revealed no significant pathology. On day 25, the patient was transferred back to the referring hospital and went home a few days later without symptoms. Outpatient follow up two months later showed that left ventricular function had improved, the cavity had remained of normal size, and no mass was visible on transthoracic echocardiography. The ECG showed persistent anterior T wave inversion.

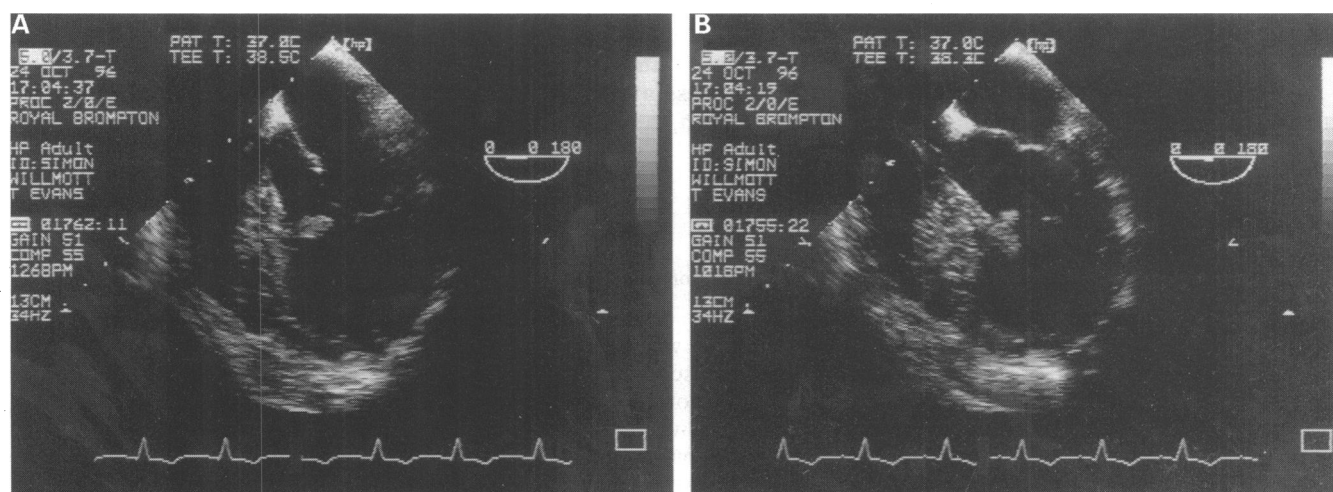


Figure 2 Transoesophageal echocardiogram in four chamber view showing a mass attached to the left ventricular side of the interventricular septum. (A) diastole; (B) systole.

Discussion

Cardiac contusion is a common injury in association with blunt chest trauma, and can involve any or all of the three layers of the heart.¹ In one series of 77 patients with blunt chest trauma 55% had regional wall motion abnormalities, the majority of which involved the right ventricle, reflecting its anterior position.² The microscopic injury seen in myocardial contusion is of a similar type to that seen in ischaemia, with oedema, white cell infiltration, loss of cellular striation and nuclear integrity, and cell necrosis, but the border between healthy and injured myocardium is less demarcated in the former condition. Severe trauma can lead to cardiac rupture and rapid death, this being the mechanism of death in 65% of fatalities in one postmortem study that included 546 cases, 106 with evidence of multichamber rupture.³ Rupture of the interventricular septum is, in fact, relatively uncommon, occurring in only 6% of the cases in this necropsy study. It is likely that the trauma experienced by the present patient resulted in a strip of myocardium being torn away from the body of the septum, as well as contusing an extensive area of the septum and apex of the left ventricular muscle. The differential diagnosis for the intracardiac mass is thrombus, which is known to be able to complicate myocardial contusion that involves the endocardial surface.

ECG abnormalities are common in cardiac contusion, most commonly T wave changes

or, less often, ST segment depression, and tend to be delayed compared with those seen in myocardial ischaemia.^{1,4,5} Localised ST segment elevation is uncommon. In the present case, the bundle branch block pattern was replaced after two to three days by anterior ST segment elevation, presumably as a result of the local contusion, although there was concern that there had been damage to the left coronary tree that was later excluded. Although there has been some improvement in left ventricular systolic function in this patient, it is likely that the healing phase of the inflammatory response involving the contused area has led to some scar formation and loss of viable myocardium, and the intention is therefore to continue with an ACE inhibitor indefinitely. Oral anticoagulation has been stopped because the risk of intracardiac thrombus seems very small, in contrast to the situation immediately after the accident.

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